Developmental Biochemistry Of the Intestine

During development, the small intestine undergoes many enzymological and physiological alterations in preparing the human fetus for extrauterine life. Glucose and 1-amino acids are actively transported by the twelfth week of fetal life. Activities of intestinal alkaline phosphatase, proteases, dipeptidases and disaccharidases are detectable in the ten-week fetus and increase rapidly after the twelfth week. Maltase and lactose increase more slowly, but are developed by the 28th week of fetal life.

Pancreatic proteolytic enzymes are active after the fifth month, and lipase activity is developed by the seventh month of fetal life. Pancreatic amylase is barely detectable at term, but increases rapidly during early post-natal life. Except for certain saturated fats and starches, the human fetus is able to digest and absorb most nutrients by the 28th week of gestation.

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Disseminated Intravascular Coagulation

Disseminated intravascular coagulation (DIC) refers to the activation of the coagulation mechanism within the circulation with deposition of fibrin in the vascular periphery. When extensive, it has two severe effects: (1) It may consume coagulation factors so rapidly that an acquired bleeding disorder develops, characterized by hypo- or afibrinogenemia (defibrination), thrombocytopenia, and low levels of factors II, V, and VIII; and (2) it may interfere with peripheral blood flow with resultant tissue damage. Severe DIC with defibrination may occur in a variety of disorders, such as bacterial sepsis, certain viral, fungal and rickettsial infections, hemolytic transfusion reactions, hemangiomas, snake bites, malignancies (neuroblastoma, leukemia), and obstetrical emergencies (placenta previa, abruptio placentae).

Laboratory tests of value in the diagnosis of DIC are prolongation of the thrombin and partial thromboplastin times, low levels of factors I (fibrinogen), II, V, VIII and platelets, and elevated levels of fibrin split products in the serum (as a result of secondary fibrinolysis of deposited fibrin). For most clinical situations, hypofibrinogenemia (fibrinogen less than 150 mg per 100 ml) and thrombocytopenia (platelets fewer than 100,000 per cu mm) are the two most characteristic laboratory abnormalities.

Treatment of severe DIC is initially aimed at removing the stimulus for coagulation by prompt treatment of the underlying disease (for example, antibiotics for infections). The decision to use heparin to prevent further fibrin deposition is dependent on: (1) The likelihood of continuing intravascular coagulation, and (2) the degree of tissue damage already present. Thus, if the etiology of the DIC is such that interruption of the process cannot be immediately accomplished (for example, in a viral disease) or if there is a grave risk of loss of soft tissue or extremities with additional thrombosis, heparin therapy should be instituted.

Other supportive therapy may include intravenous fluids for circulation support, fresh blood, fresh or fresh-frozen plasma or fibrinogen for bleeding, and low molecular weight dextran for excessive tissue destruction.

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